WEST Search History

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DATE: Monday, August 01, 2005

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		DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; PLUR=YES; OP=OR	
☐	L3	(vitro with ($absorbed or $absorb or $absorption)) same (antibody or antibodies or sera or serum or antisera or antiserum)	115
☐	L2	(vitro with $absorbed or $absorb or $absorption) same (antibody or antibodies or sera or serum or antisera or antiserum)	15752
☐	L1	(vivo with antigen) same $absor$	44

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NEWS	1		Web Page URLs for STN Seminar Schedule - N. America
NEWS	2		"Ask CAS" for self-help around the clock
NEWS	3	FEB 28	PATDPAFULL - New display fields provide for legal status data from INPADOC
NEWS	4	FEB 28	BABS - Current-awareness alerts (SDIs) available
NEWS	5	MAR 02	GBFULL: New full-text patent database on STN
NEWS	6	MAR 03	REGISTRY/ZREGISTRY - Sequence annotations enhanced
NEWS	7	MAR 03	MEDLINE file segment of TOXCENTER reloaded
NEWS	8	MAR 22	KOREAPAT now updated monthly; patent information enhanced
NEWS	9	MAR 22	Original IDE display format returns to REGISTRY/ZREGISTRY
NEWS	10	MAR 22	PATDPASPC - New patent database available
NEWS	11	MAR 22	REGISTRY/ZREGISTRY enhanced with experimental property tags
NEWS	12	APR 04	EPFULL enhanced with additional patent information and new fields
NEWS	13	APR 04	EMBASE - Database reloaded and enhanced
NEWS	14	APR 18	New CAS Information Use Policies available online
NEWS	15	APR 25	Patent searching, including current-awareness alerts (SDIs), based on application date in CA/CAplus and USPATFULL/USPAT2 may be affected by a change in filing date for U.S. applications.
NEWS	16	APR 28	Improved searching of U.S. Patent Classifications for U.S. patent records in CA/CAplus
NEWS	17	MAY 23	GBFULL enhanced with patent drawing images
NEWS	18	MAY 23	REGISTRY has been enhanced with source information from CHEMCATS
NEWS	19	JUN 06	The Analysis Edition of STN Express with Discover! (Version 8.0 for Windows) now available
NEWS	20	JUN 13	RUSSIAPAT: New full-text patent database on STN
NEWS	21	JUN 13	FRFULL enhanced with patent drawing images
NEWS	22	JUN 27	MARPAT displays enhanced with expanded G-group definitions and text labels
NEWS	23	JUL 01	MEDICONF removed from STN
NEWS	24	JUL 07	STN Patent Forums to be held in July 2005
NEWS	25	JUL 13	SCISEARCH reloaded
NEWS	26	JUL 20	Powerful new interactive analysis and visualization software, STN AnaVist, now available

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FILE 'HOME' ENTERED AT 17:31:58 ON 01 AUG 2005

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FULL ESTIMATED COST	0.21	0.21

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=> (culture or cultured or vitro) (s) cell (s) (?adsorb or ?adsorbed or ?adsorption or ?absorbed or ?absorb or ?absorption)
2 FILES SEARCHED...
L1 3028 (CULTURE OR CULTURED OR VITRO) (S) CELL (S) (?ADSORB OR ?ADSORBED OR ?ADSORPTION OR ?ABSORBED OR ?ABSORB OR ?ABSORPTION)

=> vivo (s) (antigen or epitope)
L2 26612 VIVO (S) (ANTIGEN OR EPITOPE)

=> l1 and l2
L3 32 L1 AND L2

=> dup rem l3
PROCESSING COMPLETED FOR L3
L4 21 DUP REM L3 (11 DUPLICATES REMOVED)

=> t ti l4 1-21

L4 ANSWER 1 OF 21 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
TI Biocompatible fluorescent silicon nanoparticle, useful in in-vitro and in-vivo optical imaging, comprises a fluorescent silicon nanoparticle and a biocompatible coating.

L4 ANSWER 2 OF 21 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
TI Fluorescent nanoparticles useful as pharmaceutical carriers for treating tumors, comprises a core comprising a fluorescent silane compound and a silica shell on the core.

L4 ANSWER 3 OF 21 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
TI New CD4+CD25- or Tr1-like regulatory T cells, which are able to exert contact-independent regulatory functions, useful for preparing a medicament for treating autoimmune diseases, e.g. graft rejection or graft versus host disease.

L4 ANSWER 4 OF 21 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
TI New regulatory medicament comprising CD4+CD25- T cells or Tr1-like regulatory T cells, which are able to exert contact-independent regulatory functions, useful for treating autoimmune diseases, e.g. graft rejection.

L4 ANSWER 5 OF 21 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
TI Obtaining a population of mature dendritic cells, useful for treating a disease, comprises administering an immune response modifier molecule that is an agonist of a Toll-like receptor to a subject.

L4 ANSWER 6 OF 21 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
TI New neurogenic differentiation gene, useful in gene therapy to correct traumatic neural injury that has resulted in loss of motor or sensory neural function and for constructing recombinant cell lines.

L4 ANSWER 7 OF 21 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI In vivo induced genes identified in Porphyromonas gingivalis by IVIAT.

L4 ANSWER 8 OF 21 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
TI Novel polypeptides and polynucleotides useful for diagnosing, preventing, treating neural, immune system, muscular, reproductive, pulmonary, cardiovascular, renal, proliferative disorders and cancerous diseases.

L4 ANSWER 9 OF 21 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI Differential expression of CH809 by Candida albicans during oropharyngeal candidiasis and disseminated candidiasis.

L4 ANSWER 10 OF 21 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI IVIAT screening of the entire Actinobacillus actinomycetemcomitans HK1651 genome for in vivo induced genes.

L4 ANSWER 11 OF 21 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
TI Probing for in vivo induced Porphyromonas gingivalis virulence genes using IVIAT.

L4 ANSWER 12 OF 21 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
TI Immortalised intestinal epithelial **cell** line - useful as an in **vitro** model of drug **absorption** through the gut.

L4 ANSWER 13 OF 21 MEDLINE on STN DUPLICATE 1
TI Enhanced immunogenicity of a recombinant genital warts vaccine adjuvanted with monophosphoryl lipid A.

L4 ANSWER 14 OF 21 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
TI Modified recombinant virus containing exogenous calicivirus DNA - useful in vaccines and for in vitro production of calicivirus antigens, for generation of therapeutic or diagnostic antibodies.

L4 ANSWER 15 OF 21 CAPLUS COPYRIGHT 2005 ACS on STN
TI Liver cell uptake and degradation of soluble immunoglobulin G immune complexes in vivo and in vitro in rats

L4 ANSWER 16 OF 21 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN

TI Hindered linking agents - are substd. 2-imino thiolane hydrohalide(s), used to form di sulphide linkages.

L4 ANSWER 17 OF 21 MEDLINE on STN DUPLICATE 2
TI The serologic response to Meth A sarcoma vaccines after cyclophosphamide treatment is additionally increased by various adjuvants.

L4 ANSWER 18 OF 21 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN DUPLICATE 3
TI MURINE T CELL MEDIATED CYTO TOXICITY AGAINST SYNGENEIC AND ALLOGENEIC CELL LINES INDUCED BY FETAL CALF SERUM.

L4 ANSWER 19 OF 21 MEDLINE on STN DUPLICATE 4
TI Tumor-specific antigens on rat liver cells transformed in vitro by chemical carcinogens.

L4 ANSWER 20 OF 21 MEDLINE on STN DUPLICATE 5
TI Hypersensitivity to bacteria in eczema. IV. Cytotoxic effect of antibacterial antibody on skin cells acquiring bacterial antigens.

L4 ANSWER 21 OF 21 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
TI Preparation and properties of antisera directed against antigens of the P 815 mastocytoma cell not shared by its syngeneic host, the DBA/2 mouse.

=> d ibib abs l4 1-11, 13-21

L4 ANSWER 1 OF 21 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
ACCESSION NUMBER: 2005-048581 [05] WPIDS
DOC. NO. CPI: C2005-016654
TITLE: Biocompatible fluorescent silicon nanoparticle, useful in in-vitro and in-vivo optical imaging, comprises a fluorescent silicon nanoparticle and a biocompatible coating.
DERWENT CLASS: A96 B04 D16
INVENTOR(S): GROVES, K; MADDEN, K N; POSS, K G; RAJOPADHYE, M
PATENT ASSIGNEE(S): (VISE-N) VISEN MEDICAL INC
COUNTRY COUNT: 108
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2004108902	A2	20041216	(200505)*	EN	58

RW: AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NA NL OA PL PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NA NI NO NZ OM PG PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2004108902	A2	WO 2004-US18023	20040604

PRIORITY APPLN. INFO: US 2003-475802P 20030604
AN 2005-048581 [05] WPIDS
AB WO2004108902 A UPAB: 20050124

NOVELTY - Biocompatible fluorescent silicon nanoparticle (A) comprises a fluorescent silicon nanoparticle (1) and a biocompatible coating (2).

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for:

(1) an in vitro optical imaging method comprising contacting a sample with fluorescent silicon nanoparticle imaging probes (Aa); allowing (Aa) to become activated or bind to the biological target of interest in the sample; optionally, removing the unbound (Aa); illuminating the target with light of a wavelength absorbable by (Aa); and detecting the optical signal emitted by (Aa) and

(2) a method of in vivo optical imaging comprising:

(a) administering to a subject (Aa);

(b) allowing (a) to contact a biological target;

(c) illuminating the target with light of a wavelength absorbable by (a); and

(d) detecting the optical signal emitted by (a).

USE - (A) is useful in in-vivo and in-vitro optical imaging, which are useful in the early detection or staging of a disease; in monitoring or determining a therapeutic course of action (surgical or administration of a drug therapy) for a treatment of a disease; or in assessing the effect of one or more drug therapies on a disease state (cancer, cardiovascular diseases, neurodegenerative diseases, immunologic diseases, autoimmune diseases, metabolic diseases, inherited diseases, infectious diseases, bone diseases or environmental diseases) (claimed).

ADVANTAGE - (A) is biocompatible, non-immunogenic, nontoxic, and can be derivatized or conjugated with affinity ligands e.g. biological or targeting moieties.

Dwg.0/4

L4 ANSWER 2 OF 21 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN

ACCESSION NUMBER: 2004-399669 [37] WPIDS

DOC. NO. CPI: C2004-149483

TITLE: Fluorescent nanoparticles useful as pharmaceutical carriers for treating tumors, comprises a core comprising a fluorescent silane compound and a silica shell on the core.

DERWENT CLASS: B04 D16

INVENTOR(S): OW, H; WIESNER, U; WIESNER, U B; LARSON, D E; WEBB, W W

PATENT ASSIGNEE(S): (OWHH-I) OW H; (WIES-I) WIESNER U; (CORR) CORNELL RES FOUND INC; (WIES-I) WIESNER U B; (LARS-I) LARSON D E; (WEBB-I) WEBB W W

COUNTRY COUNT: 107

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 2004101822	A1	20040527	(200437)*		21
WO 2004063387	A2	20040729	(200451)	EN	
RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NI NO NZ OM PG PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW					
WO 2004074504	A2	20040902	(200457)	EN	
RW: AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NI NO NZ OM PG PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW					

AU 2003303309 A1 20040810 (200479)
AU 2003303290 A1 20040909 (200501)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 2004101822	A1	US 2002-306614	20021126
WO 2004063387	A2	WO 2003-US37793	20031126
WO 2004074504	A2	WO 2003-US37963	20031126
AU 2003303309	A1	AU 2003-303309	20031126
AU 2003303290	A1	AU 2003-303290	20031126

FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2003303309	A1 Based on	WO 2004063387
AU 2003303290	A1 Based on	WO 2004074504

PRIORITY APPLN. INFO: US 2002-306614 20021126
AN 2004-399669 [37] WPIDS
AB US2004101822 A UPAB: 20040611
NOVELTY - A fluorescent nanoparticle (I) comprising a core comprising a fluorescent silane compound, and a silica shell on the core, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for:

(1) preparing (M1), (I) involves mixing a fluorescent compound and an organo-silane compound to form a fluorescent core, and mixing the resulting core with a silica forming compound to form a silica shell on the core, to provide the fluorescent nanoparticle;

(2) monitoring (M2) movement of a cellular component of a cell, involves contacting the cell with a ligated-fluorescent nanoparticle to form a cell selectively labelled with the ligated-fluorescent nanoparticle and to form a fluorescent loci and recording the motion of the fluorescent loci for a time, to monitor the movement of a cellular component;

(3) a pharmaceutical carrier comprising (I) and optionally a ligand;

(4) a pharmaceutical composition comprising (I) and optionally a therapeutic agent;

(5) an imaging agent comprising (I);

(6) treating (M3) disease or disorder, comprising:

(a) administering to a patient in need of treatment a ligated-fluorescent nanoparticle optionally including a therapeutic agent, the nanoparticle being adapted to selectively associate with a disease producing component of the cell, to form a selectively decorated cell with the ligated-fluorescent nanoparticle, and illuminating the decorated cell to treat disease or disorder; or

(b) contacting a cell with a ligated-fluorescent nanoparticle to form a cell selectively decorated with the ligated-fluorescent nanoparticle, and irradiating the resulting decorated cell to treat the disease or disorder;

(7) a kit for use in the detection of an analyte, the kit comprising packaging material containing a ligated-fluorescent nanoparticle;

(8) a kit for detecting and monitoring a cell surface component, the kit comprising packaging material containing a ligated-fluorescent nanoparticle for detecting the cell surface component, and optionally a recorder for monitoring the cell surface component;

(9) detecting (M4) motion or a change in the location of a cellular component of a cell when the cell is treated with a therapeutic agent, involves contacting a cell with a ligated-fluorescent nanoparticles, the nanoparticle having a therapeutic agent, to bind the ligated-fluorescent nanoparticle to a cellular component, and recording fluorescent signal, to detect the motion or location change of the component; and

(10) detecting the presence of an analyte, involves contacting a sample which may contain an analyte with a ligated-fluorescent nanoparticles adapted to associate with the analyte, if present, to form a ligated-fluorescent nanoparticle-analyte complex, optionally separating uncomplexed ligated-fluorescent nanoparticles, and detecting the fluorescent signal of ligated-fluorescent nanoparticle-analyte complex to establish the presence of the analyte.

ACTIVITY - Cytostatic.

No biological data given.

MECHANISM OF ACTION - None given.

USE - (I) is useful for monitoring movement of a cellular component of a cell. (I) is useful as a pharmaceutical composition and as an imaging agent. (I) is useful for detecting motion or change in location of a cellular component of a cell and for detecting the presence of an analyte (I) is also useful as a pharmaceutical carrier, for treating a disease or disorder, where the disease is cancerous tumor. The disease is sensitive to fluorescence, heat or both (claimed).

(I) is useful in diagnostic kits or assays such as immuno assays, in improved imaging agents, in purification process, drugs e.g., treatment regimens and therapies such as drug delivery to specifically target and shrink tumors or to identify and separate infectious agents etc.

Dwg.0/0

L4 ANSWER 3 OF 21 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN

ACCESSION NUMBER: 2004-182275 [18] WPIDS
CROSS REFERENCE: 2004-182226 [18]
DOC. NO. CPI: C2004-072121
TITLE: New CD4+CD25- or Tr1-like regulatory T cells, which are able to exert contact-independent regulatory functions, useful for preparing a medicament for treating autoimmune diseases, e.g. graft rejection or graft versus host disease.
DERWENT CLASS: B04 D16
INVENTOR(S): DIECKMANN, D; SCHULER, G
PATENT ASSIGNEE(S): (SCHU-I) SCHULER G; (DIEC-I) DIECKMANN D
COUNTRY COUNT: 31
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
EP 1391504	A1	20040225	(200418)*	EN	17
R: AL AT BE BG CH CY CZ DE DK EE ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI SK TR					
US 2004147021	A1	20040729	(200450)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
EP 1391504	A1	EP 2002-18025	20020812
US 2004147021	A1 Provisional	US 2002-419177P	20021017
		US 2003-618134	20030711

PRIORITY APPLN. INFO: EP 2002-18025 20020812
AN 2004-182275 [18] WPIDS
CR 2004-182226 [18]
AB EP 1391504 A UPAB: 20040805
NOVELTY - CD4+CD25- T cells or Tr1-like regulatory T cells which are able to exert contact-independent regulatory functions, are new. The Tr1-like regulatory T cells are obtainable by anergizing the CD4+CD25- T cells, preferably by contact with CD4+CD25+ T cells.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) expanding CD4+CD25- T cells or TR1-like regulatory T cells by stimulating the T cells with a T cell stimulating agent or with **antigen**-presenting cells ex **vivo** and in **vivo**;

(2) producing the TR1-like regulatory T cells;

(3) a pharmaceutical composition comprising the human CD4+CD25- T cells or TR1-like regulatory T cells;

(4) adoptive transfer therapy comprising injecting/infusing back into the patients enriched/expanded autologous or non-autologous TR1-like regulatory T cells;

(5) preparing CD4+CD25- T cells and TR1-like regulatory T cells with a particular desired antigen-specific T cell receptor;

(6) expanded CD4+CD25- T cells and TR1-like regulatory T cells, or obtainable from the methods above;

(7) CD4+CD25- T cells and TR1-like regulatory T cells having a particular desired **antigen**-specific T cell receptor and obtained by using the method of (5) or by transfection of a T cell receptor of desired **antigen** specificity into ex **vivo** isolated or expanded T cells or using the method of (5), and which have been brought in anergic state; and

(8) a pharmaceutical composition comprising the T cells, for treating diseases with enhanced immunity including, but not limited to, autoimmune diseases, graft versus host disease, and graft rejection.

ACTIVITY - Immunosuppressive.

No biological data given.

MECHANISM OF ACTION - Cellular therapy.

USE - The CD4+CD25- T cells or of TR1-like regulatory T cells are useful for preparing a regulatory medicament, in assays that will allow to identify other regulatory factors, for identifying molecules expressed by the CD4+CD25- T cells or by the TR1-like regulatory T cells including identification of novel molecules on the cells, for identifying precursor cells or progeny of the regulatory CD4+CD25- T cells or of the TR1-like regulatory T cells, and for preparing an agent for adoptive transfer therapy, an agent for treating diseases with enhanced immunity including but not limited to autoimmune diseases, or an agent for preventing/treating transplantation reactions such as graft versus host disease, graft rejections, etc. The anergic state-inducing agent can be used for preparing an agent to induce TR1-like regulatory T cells in vivo, particularly for preparing an agent for treating autoimmune diseases in a patient. Agents specifically binding to defined entities on the TR1-like regulatory T cells, including but not limited to ligands/antibodies, such as monoclonal antibodies or MHC-peptide complexes or other ligands binding to T cell receptors on (subsets of) the TR1-like regulatory T cells, for preparing a medicament for the removal or functional impairment of TR1-like regulatory T cells in vivo to enhance immune responses, including dampen regulation by and Tr1-like regulatory T cells in vivo, e.g., to enhance tumor immunity (all claimed).

Dwg.0/4

L4 ANSWER 4 OF 21 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN

ACCESSION NUMBER:	2004-182226 [18] WPIDS
CROSS REFERENCE:	2004-182275 [18]
DOC. NO. CPI:	C2004-072106
TITLE:	New regulatory medicament comprising CD4+CD25- T cells or Tr1-like regulatory T cells, which are able to exert contact-independent regulatory functions, useful for treating autoimmune diseases, e.g. graft rejection.
DERWENT CLASS:	B04 D16
INVENTOR(S):	DIECKMANN, D; SCHULER, G
PATENT ASSIGNEE(S):	(SCHU-I) SCHULER G
COUNTRY COUNT:	31

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
EP 1391210	A2	20040225	(200418)*	EN	18

R: AL AT BE BG CH CY CZ DE DK EE ES FI FR GB GR HU IE IT LI LT LU LV MC MK NL PT RO SE SI SK TR

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
EP 1391210	A2	EP 2003-102508	20030812

PRIORITY APPLN. INFO: EP 2002-18025 20020812
AN 2004-182226 [18] WPIDS
CR 2004-182275 [18]
AB EP 1391210 A UPAB: 20040316

NOVELTY - A regulatory medicament comprising CD4+CD25- T cells or Tr1-like regulatory T cells, which are able to exert contact- independent regulatory functions, is new.

DETAILED DESCRIPTION - A regulatory medicament comprising CD4+CD25- T cells or Tr1-like regulatory T cells, which are able to exert contact-independent regulatory functions, is new. The Tr1-like regulatory T cells are obtainable by anergizing the CD4+CD25- T cells, preferably by contact with CD4+CD25+ T cells.

INDEPENDENT CLAIMS are also included for:

(1) preparing CD4+CD25- T cells and TR1-like regulatory T cells with a particular desired antigen-specific T cell receptor;

(2) CD4+CD25- T cells and TR1-like regulatory T cells having a particular desired antigen-specific T cell receptor and obtained by:

(a) using the method above or by transfection of a T cell receptor of desired **antigen** specificity into ex **vivo** isolated or expanded T cells; or

(b) using the method above, and which have been brought in anergic state; and

(3) a pharmaceutical composition comprising the T cells of (2), for treating diseases with enhanced immunity including, but not limited to, autoimmune diseases, graft versus host disease and graft rejections.

ACTIVITY - Immunosuppressive.

No biological data given.

MECHANISM OF ACTION - Cellular Therapy.

USE - The CD4+CD25- T cells or of TR1-like regulatory T cells are useful for preparing a regulatory medicament, in assays that will allow to identify other regulatory factors, for identifying molecules expressed by the CD4+CD25- T cells or by the TR1-like regulatory T cells including identification of novel molecules on the cells, for identifying precursor cells or progeny of the regulatory CD4+CD25- T cells or of the TR1-like regulatory T cells, and for preparing an agent for adoptive transfer therapy, an agent for treating diseases with enhanced immunity including but not limited to autoimmune diseases, or an agent for preventing/treating transplantation reactions such as graft versus host disease, graft rejections, etc. The anergic state-inducing agent can be used for preparing an agent to induce TR1-like regulatory T cells in vivo, particularly for preparing an agent for treating autoimmune diseases in a patient. Agents specifically binding to defined entities on the TR1-like regulatory T cells, including but not limited to ligands/antibodies, such as monoclonal antibodies or MHC-peptide complexes or other ligands binding to T cell receptors on (subsets of) the TR1-like regulatory T cells, for preparing a medicament for the removal or functional impairment of TR1-like regulatory T cells in vivo to enhance immune responses, including

dampen regulation by and Tr1-like regulatory T cells in vivo, e.g., to enhance tumor immunity (all claimed).
Dwg.0/7

L4 ANSWER 5 OF 21 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
ACCESSION NUMBER: 2003-393260 [37] WPIDS
DOC. NO. CPI: C2003-104375
TITLE: Obtaining a population of mature dendritic cells, useful for treating a disease, comprises administering an immune response modifier molecule that is an agonist of a Toll-like receptor to a subject.
DERWENT CLASS: B04 D16
INVENTOR(S): STOLPA, J C; TOMAI, M A; VASILAKOS, J P
PATENT ASSIGNEE(S): (MINN) 3M INNOVATIVE PROPERTIES CO
COUNTRY COUNT: 102
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2003020889	A2	20030313	(200337)*	EN	84
RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SK SL SZ TR TZ UG ZM ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SD SE SG SI SK SL TJ TM TN TR TT TZ UA UG UZ VC VN YU ZA ZM ZW					
US 2003133913	A1	20030717	(200348)		
EP 1427445	A2	20040616	(200439)	EN	
R: AL AT BE BG CH CY CZ DE DK EE ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI SK TR					
AU 2002329892	A1	20030318	(200452)		
JP 2005501550	W	20050120	(200508)		143
IN 2004000453	P4	20041218	(200533)		

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 2003020889	A2		WO 2002-US27393	20020828
US 2003133913	A1	Provisional	US 2001-316144P	20010830
		Provisional	US 2002-370177P	20020405
			US 2002-229829	20020828
EP 1427445	A2		EP 2002-766145	20020828
			WO 2002-US27393	20020828
AU 2002329892	A1		AU 2002-329892	20020828
JP 2005501550	W		WO 2002-US27393	20020828
			JP 2003-525593	20020828
IN 2004000453	P4		WO 2002-US27393	20020828
			IN 2004-CN453	20040301

FILING DETAILS:

PATENT NO	KIND		PATENT NO
EP 1427445	A2	Based on	WO 2003020889
AU 2002329892	A1	Based on	WO 2003020889
JP 2005501550	W	Based on	WO 2003020889

PRIORITY APPLN. INFO: US 2002-370177P 20020405; US 2001-316144P 20010830; US 2002-229829 20020828

AN 2003-393260 [37] WPIDS

AB WO2003020889 A UPAB: 20030612

NOVELTY - Obtaining (M1) a population of mature dendritic cells, comprises administering an immune response modifier molecule (IRM) that is an agonist of Toll-like receptor (TLR)-6, TLR-7, or TLR-8 to a subject in an amount effective to mature dendritic cells of the subject, and isolating the mature dendritic cells.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) a cell population (I) obtained by (M1);

(2) enhancing (M2) antigen presentation by dendritic cells in vitro, comprising:

(a) exposing an isolated dendritic cell population to an antigen;

(b) contacting the isolated dendritic cell with IRM; and

(c) allowing the dendritic cell to process and present the antigen;

(3) an isolated dendritic cell population (II) produced by (M2);

(4) detecting (M3) cytokine production, expression of co-stimulatory markers, or expression of chemokine receptors by a plasmacytoid dendritic cell (pDC), comprising:

(a) contacting isolated pDC with IRM for inducing the plasmacytoid dendritic cell to produce one or more cytokines selected from interleukin (IL)-8, IP-10, IL-6, macrophage Inflammatory Protein 1 alpha (MIP-1 alpha), and interferon (IFN)- omega , or to express one or more co-stimulatory marker or chemokine receptor; and

(b) detecting production of one of the cytokines, co-stimulatory marker, or chemokine receptor by the dendritic cell;

(5) enhancing (M4) survival of isolated plasmacytoid dendritic cells, comprising:

(a) contacting a population of isolated pDCs with an IRM in an amount effective for enhancing survival of the pDCs; and

(b) incubating pDCs under conditions so that 30 % of pDC survive for 48 hours;

(6) identifying (M5) a compound that selectively induces production of a chemokine receptor by pDCs, comprising:

(a) obtaining a population of cells that includes both inflammatory cytokine producing cells and pDCs;

(b) contacting the population of cells with a test compound;

(c) determining the amount of chemokine receptor present in the population of cells contacted with the test compound;

(d) determining the amount of inflammatory cytokine(s) present in the population of cells contacted with the test compound; and

(e) identifying the test compound as a selective inducer of the chemokine receptor if the chemokine receptor is present in the population of cells after contact with the test compound in an amount 3 times greater than the amount of inflammatory cytokine(s) present in the population of cells;

(7) preparing (M6) a cell population enriched for cells that express a chemokine receptor, comprising:

(a) contacting pDC with IRM for inducing pDC to express one or more chemokine receptor; and

(b) enriching the cell population for cells that express a chemokine receptor;

(8) a population of pDCs enriched for cells that express chemokine receptors prepared by (M6); and

(9) a cellular adjuvant (III) prepared by maturing pDCs in vitro by treating dendritic cells with IRM, and exposing mature pDCs to antigens associated with the disease.

ACTIVITY - Neuroprotective; Antidiabetic; Antirheumatic; Antiarthritic; Antiinflammatory; Antipsoriatic; Tuberculostatic; Antileprotic; Protozoacide; Fungicide; Virucide; Antiparasitic; Antibacterial; Dermatological; Antiallergic; Anti-HIV; Antiasthmatic; Immunosuppressive; Nootropic; Cardiant; Cytostatic; Muscular;

Immunostimulant.

MECHANISM OF ACTION - Ex vivo gene therapy; Vaccine. No biological data is given.

USE - (M1) And another new method (M2) are useful for treating a disease, by:

(a) contacting an isolated pDC with IRM for inducing pDC to express chemokine receptors;

(b) contacting the population of pDC with an antigen associated with the disease;

(c) enriching the cell population for cells expressing a high level of a chemokine receptor; and

(d) administering the enriched cell population to a patient.

A cell population (I) obtained by (M1), or a cellular adjuvant (III) is useful for treating a disease. (M1) Is useful for preparing a cellular adjuvant for the treatment of a disease, by maturing pDCs in vitro, and exposing mature pDCs to antigens associated with the disease. The disease is a neoplastic disease and the antigen is derived from neoplastic cells. The disease is caused by an infectious agent and the antigen is derived from the infectious agent. The disease is a Th2-mediated disease (claimed). (I) Is useful for enhancing antigen presenting ability, to enhance the immune response of a subject, in immunotherapies, ex vivo cell transplantation therapies for treating e.g., acquired immunodeficiency syndrome (AIDS), in ex vivo expansion of T-cells which are useful for treat disorders characterized by deterioration of immune system, for the generation of monoclonal antibodies that recognize pDC-specific markers, for the preparation of antigen-activated pDCs, and for the development of vaccines and vaccine adjuvants. (III) Is useful for provoking an anti-tumor immune response in the patient, for the treatment of non-infections protein-related disease e.g., Alzheimer's disease, and heart disease. (I) Is useful to produce cytokines that favor the generation of Th1 immune response, for treating asthma, allergic rhinitis, systemic lupus erythematosis, eczema, atopic dermatitis, parasitic infections e.g., cutaneous and systemic leishmaniais, fungal infection e.g., candidiasis and histoplasmosis, and intracellular bacterial infection e.g., leprosy and tuberculosis, for the treatment of disorders mediated by T-cells e.g., psoriasis, inflammatory bowel disease, rheumatoid arthritis, diabetes, multiple sclerosis, and other diseases associated with chronic T-cells activation.

Dwg.0/5

L4 ANSWER 6 OF 21 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN

ACCESSION NUMBER: 2003-056678 [05] WPIDS

CROSS REFERENCE: 1995-404081 [51]; 1997-272117 [24]; 1998-466661 [40]

DOC. NO. CPI: C2003-014491

TITLE: New neurogenic differentiation gene, useful in gene therapy to correct traumatic neural injury that has resulted in loss of motor or sensory neural function and for constructing recombinant cell lines.

DERWENT CLASS: B04 D16

INVENTOR(S): TAPSCOTT, S J

PATENT ASSIGNEE(S): (HUTC-N) HUTCHINSON CANCER RES CENT FRED

COUNTRY COUNT: 1

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 6444463	B1	20020903	(200305)*		43

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE

US 6444463	B1	CIP of	US 1994-239238	19940506
		CIP of	WO 1995-US5741	19950508
		CIP of	US 1995-552142	19951102
		CIP of	WO 1996-US17532	19961030
		CIP of	US 1997-910973	19970807
		Cont of	WO 1998-US16417	19980805
			US 2000-499227	20000207

FILING DETAILS:

PATENT NO	KIND		PATENT NO
US 6444463	B1	CIP of	US 5695995
		CIP of	US 5795723

PRIORITY APPLN. INFO: WO 1998-US16417 19980805; US 1994-239238 19940506; WO 1995-US5741 19950508; US 1995-552142 19951102; WO 1996-US17532 19961030; US 1997-910973 19970807; US 2000-499227 20000207

AN 2003-056678 [05] WPIDS

CR 1995-404081 [51]; 1997-272117 [24]; 1998-466661 [40]

AB US 6444463 B UPAB: 20030121

NOVELTY - An isolated nucleic acid molecule (I) which hybridizes under stringent conditions with a nucleic acid sequence of 1268 bp given in the specification, and which encodes a functionally active neuroD3 polypeptide, or a nucleic acid molecule which encodes a human neuroD3 polypeptide having the sequence of 237 amino acids given in the specification, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) a vector comprising in serial array, a promoter, and (I); and

(2) a cell (II) in culture transformed by (I).

ACTIVITY - Neuroprotective; Auditory; Ophthalmological. No supporting data is given.

MECHANISM OF ACTION - Gene therapy. No supporting data is given.

USE - (I) is useful for constructing recombinant **cell** lines, ova, and transgenic embryos and animals including dominant-negative and knock-out recombinant **cell** lines, cDNA and oligonucleotide probes for Northern or Southern blots, or polymerase chain reaction (PCR) assays for identifying and quantifying the level of expression of neuroD in a **cell**. Birth defects and spontaneous abortions may result from expression of an abnormal neuroD protein, thus screening neuroD expression may be useful in prenatal screening of mothers in utero. NeuroD gene therapy may be used to correct traumatic neural injury that has resulted in loss of motor or sensory neural function. The neuroD genes are also useful for preparation of transplantable recombinant neuronal precursor **cell** populations from embryonic ectodermal cells, and non-neural basal stem cells. The polynucleotide sequences permit the establishment of primary cultures of proliferating embryonic neuronal stem cells under conditions mimicking those that are active in development and cancer. The resultant **cell** lines find use as sources of novel neural growth factors, and in assays for identifying novel neuronal growth factors. (II) is useful in **vitro** as convenient sources of neuronal and other growth factors for screening anti-cancer drugs capable of driving terminal differentiation in neural tumors, as sources of recombinantly expressed neuroD protein for use as an **antigen** in preparing monoclonal and polyclonal antibodies useful in diagnostic assays, and for screening for compounds capable of increasing or decreasing the activity of neuroD. Transformed host cells are also useful

in **vivo** for transplantation at sites of traumatic neural injury where motor or sensory neural activity has been lost, e.g., for treating patients with hearing or vision loss due to optical or auditory nerve damage, patients with peripheral nerve damage and loss of motor or sensory neural activity, and patients with brain or spinal cord damage from traumatic injury or radiation injury. The host cells find use in the treatment of **malabsorption** syndromes or gastrointestinal dysmotility syndromes (Hirsh Prung's Disease). The **cell** lines also find use in screening for candidate therapeutic agents capable of either substituting for neuroD or correcting the cellular defect caused by a defective neuroD. **Cell** lines expressing wild-type neuroD proteins may be useful for correcting birth defects that result from defective neuroD expression.
Dwg.0/0

L4 ANSWER 7 OF 21 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 2002:570978 BIOSIS
DOCUMENT NUMBER: PREV200200570978
TITLE: In vivo induced genes identified in Porphyromonas gingivalis by IVIAT.
AUTHOR(S): Walters, S. M. [Reprint author]; Handfield, M. [Reprint author]; Hillman, J. D. [Reprint author]; Progulske-Fox, A. [Reprint author]
CORPORATE SOURCE: University of Florida, Gainesville, FL, USA
SOURCE: Abstracts of the General Meeting of the American Society for Microbiology, (2002) Vol. 102, pp. 57-58. print. Meeting Info.: 102nd General Meeting of the American Society for Microbiology. Salt Lake City, UT, USA. May 19-23, 2002. American Society for Microbiology. ISSN: 1060-2011.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 7 Nov 2002
Last Updated on STN: 7 Nov 2002

AB Periodontitis affects at least 35% of adults aged 30-90. Porphyromonas gingivalis is recognized as an important species in the pathogenesis of this disease. Additionally, epidemiological data has suggested a strong association between periodontitis and coronary heart disease (CHD). We have used In **Vivo** Induced **Antigen** Technology (IVIAT) to identify in **vivo** induced genes of two strains of Porphyromonas gingivalis. In this study serum was collected from 18 patients with active periodontitis and from whom P. gingivalis could be cultured. The sera were pooled and repeatedly **adsorbed** with in **vitro** grown P. gingivalis whole cells and **cell** extracts to remove antibodies against in **vitro** induced antigens. Genomic expression libraries of P. gingivalis strains W83 and 381 were constructed using hydrosheared DNA. The W83 library was then probed with the pooled, adsorbed patient sera. 135,000 W83 clones have been screened, and of these, 392 were identified as putative positives. The 392 clones were then screened by secondary dot blot and 130 were confirmed as positive and sequenced. Sequence analysis revealed sequences with homology to known virulence factors of P. gingivalis, sequences with homology to known genes in other organisms and sequences with homology to hypothetical genes or genes of unknown function. These potential virulence factors will be cloned and their protein products purified. Antibodies will be generated against these purified proteins and will be used to probe patient plaque samples to confirm the expression of these factors in vivo. Those that are confirmed are expected to include new classes of previously unknown virulence factors of P. gingivalis that may be useful targets for new diagnostics, antimicrobials and vaccines.

L4 ANSWER 8 OF 21 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN

ACCESSION NUMBER: 2001-465566 [50] WPIDS

CROSS REFERENCE: 2001-451924 [48]; 2001-451925 [48]; 2001-451926 [48]; 2001-451927 [48]; 2001-451928 [48]; 2001-451929 [48]; 2001-451930 [48]; 2001-451931 [48]; 2001-451932 [48]; 2001-451936 [48]; 2001-451937 [48]; 2001-457716 [49]; 2001-457717 [49]; 2001-457723 [49]; 2001-457724 [49]; 2001-457725 [49]; 2001-457726 [49]; 2001-457727 [49]; 2001-457728 [49]; 2001-465460 [50]; 2001-465557 [50]; 2001-465558 [50]; 2001-465559 [50]; 2001-465560 [50]; 2001-465565 [50]; 2001-465567 [50]; 2001-465568 [50]; 2001-465569 [50]; 2001-465570 [50]; 2001-465572 [50]; 2001-465573 [50]; 2001-465577 [50]; 2001-476159 [51]; 2001-476160 [51]; 2001-476161 [51]; 2001-476181 [51]; 2001-476182 [51]; 2001-476195 [51]; 2001-476208 [51]; 2001-476220 [51]; 2001-476222 [51]; 2001-476223 [51]; 2001-476224 [51]; 2001-476225 [51]; 2001-483227 [52]; 2001-483232 [52]; 2001-483426 [52]; 2001-488743 [53]; 2001-488776 [53]; 2001-488777 [53]; 2001-488781 [53]; 2001-488782 [53]; 2001-488783 [53]; 2001-488784 [53]; 2001-488785 [53]; 2001-488786 [53]; 2001-488787 [53]; 2001-496846 [54]; 2001-502629 [55]; 2001-502630 [55]; 2001-502866 [55]; 2001-514652 [56]; 2001-530113 [58]; 2001-541497 [60]; 2001-541565 [60]; 2001-565185 [63]; 2001-565190 [63]; 2001-581633 [65]; 2001-611720 [70]; 2001-639119 [73]; 2002-122018 [16]; 2002-147878 [19]; 2002-257198 [30]; 2002-381944 [41]; 2002-405050 [43]; 2002-453715 [48]; 2002-470713 [50]; 2002-489586 [52]; 2002-608160 [65]; 2002-635684 [68]; 2002-642242 [69]; 2002-642253 [69]; 2002-642377 [69]; 2002-665432 [71]; 2002-681727 [73]; 2002-690611 [74]; 2002-705875 [76]; 2002-731367 [79]; 2003-128199 [12]; 2003-147444 [14]; 2003-174087 [17]; 2003-182526 [18]; 2003-198289 [19]; 2003-219994 [21]; 2003-265788 [26]; 2003-311001 [30]; 2003-416807 [39]; 2003-447703 [42]; 2003-447704 [42]; 2003-492122 [46]; 2003-512305 [48]; 2003-605749 [57]; 2003-605750 [57]; 2003-615767 [58]; 2003-615993 [58]; 2003-625420 [59]; 2003-634869 [60]; 2003-634870 [60]; 2003-695890 [66]; 2003-695900 [66]; 2003-708342 [67]; 2003-708345 [67]; 2003-719985 [68]; 2003-743747 [70]; 2003-743765 [70]; 2003-743766 [70]; 2003-765398 [72]; 2003-765402 [72]; 2003-765403 [72]; 2003-765488 [72]; 2003-786903 [74]; 2003-786918 [74]; 2003-787333 [74]; 2003-801167 [75]; 2003-801192 [75]; 2003-829398 [77]; 2003-901052 [82]; 2003-902033 [82]; 2004-080168 [08]; 2004-081713 [08]; 2004-090458 [09]; 2004-108205 [11]; 2004-122079 [12]; 2004-141549 [14]

DOC. NO. CPI: C2001-140550

TITLE: Novel polypeptides and polynucleotides useful for diagnosing, preventing, treating neural, immune system, muscular, reproductive, pulmonary, cardiovascular, renal, proliferative disorders and cancerous diseases.

DERWENT CLASS: B04 D16

INVENTOR(S): BARASH, S C; ROSEN, C A; RUBEN, S M

PATENT ASSIGNEE(S): (HUMA-N) HUMAN GENOME SCI INC

COUNTRY COUNT: 94

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001055301	A2	20010802	(200150)*	EN	

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ

NL OA PT SD SE SL SZ TR TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM
DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC
LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE
SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
AU 2001052878 A 20010807 (200174)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001055301	A2	WO 2001-US1239	20010117
AU 2001052878	A	AU 2001-52878	20010117

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2001052878	A	Based on	WO 2001055301

PRIORITY APPLN. INFO: US 2001-259678P 20010105; US
2000-179065P 20000131; US
2000-180628P 20000204; US
2000-184664P 20000224; US
2000-186350P 20000302; US
2000-189874P 20000316; US
2000-190076P 20000317; US
2000-198123P 20000418; US
2000-205515P 20000519; US
2000-209467P 20000607; US
2000-214886P 20000628; US
2000-215135P 20000630; US
2000-216647P 20000707; US
2000-216880P 20000707; US
2000-217487P 20000711; US
2000-217496P 20000711; US
2000-218290P 20000714; US
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2000-250160P 20001201; US
2000-250391P 20001201; US
2000-251030P 20001205; US
2000-251988P 20001205; US
2000-256719P 20001205; US
2000-251479P 20001206; US
2000-251856P 20001208; US
2000-251868P 20001208; US
2000-251869P 20001208; US
2000-251989P 20001208; US
2000-251990P 20001208; US
2000-254097P 20001211

AN 2001-465566 [50] WPIDS

CR 2001-451924 [48]; 2001-451925 [48]; 2001-451926 [48]; 2001-451927 [48];
2001-451928 [48]; 2001-451929 [48]; 2001-451930 [48]; 2001-451931 [48];
2001-451932 [48]; 2001-451936 [48]; 2001-451937 [48]; 2001-457716 [49];
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2001-465570 [50]; 2001-465572 [50]; 2001-465573 [50]; 2001-465577 [50];
2001-476159 [51]; 2001-476160 [51]; 2001-476161 [51]; 2001-476181 [51];
2001-476182 [51]; 2001-476195 [51]; 2001-476208 [51]; 2001-476220 [51];
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2001-488776 [53]; 2001-488777 [53]; 2001-488781 [53]; 2001-488782 [53];
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2001-541565 [60]; 2001-565185 [63]; 2001-565190 [63]; 2001-581633 [65];
2001-611720 [70]; 2001-639119 [73]; 2002-122018 [16]; 2002-147878 [19];
2002-257198 [30]; 2002-381944 [41]; 2002-405050 [43]; 2002-453715 [48];
2002-470713 [50]; 2002-489586 [52]; 2002-608160 [65]; 2002-635684 [68];
2002-642242 [69]; 2002-642253 [69]; 2002-642377 [69]; 2002-665432 [71];
2002-681727 [73]; 2002-690611 [74]; 2002-705875 [76]; 2002-731367 [79];
2003-128199 [12]; 2003-147444 [14]; 2003-174087 [17]; 2003-182526 [18];
2003-198289 [19]; 2003-219994 [21]; 2003-265788 [26]; 2003-311001 [30];
2003-416807 [39]; 2003-447703 [42]; 2003-447704 [42]; 2003-492122 [46];
2003-512305 [48]; 2003-605749 [57]; 2003-605750 [57]; 2003-615767 [58];
2003-615993 [58]; 2003-625420 [59]; 2003-634869 [60]; 2003-634870 [60];
2003-695890 [66]; 2003-695900 [66]; 2003-708342 [67]; 2003-708345 [67];
2003-719985 [68]; 2003-743747 [70]; 2003-743765 [70]; 2003-743766 [70];

2003-765398 [72]; 2003-765402 [72]; 2003-765403 [72]; 2003-765488 [72]; 2003-786903 [74]; 2003-786918 [74]; 2003-787333 [74]; 2003-801167 [75]; 2003-801192 [75]; 2003-829398 [77]; 2003-901052 [82]; 2003-902033 [82]; 2004-080168 [08]; 2004-081713 [08]; 2004-090458 [09]; 2004-108205 [11]; 2004-122079 [12]; 2004-141549 [14]

AB WO 200155301 A UPAB: 20040226

NOVELTY - An isolated polypeptide (I) comprising an amino acid sequence at least 90% identical to a polypeptide fragment of a sequence chosen from 640 sequences of specific amino acids (or its domain, epitope, full length protein, a variant, an allelic variant or a species homolog) or the encoded sequence contained in specific cDNA clones (given in the specification), is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) an isolated nucleic acid molecule (II) comprising a polynucleotide having a nucleotide sequence at least 95% identical to a PN fragment chosen from 640 sequences of specific nucleotides (sequence listing not provided in the specification), a PN fragment of cDNA sequence contained in specific clones (given in the specification), a PN encoding (I), a PN which is a variant, allelic variant of (II) or a PN capable of hybridizing to (II) (but does not hybridize under stringent conditions to a nucleic acid having a nucleotide sequence of only A or T residues);

(2) a recombinant vector comprising (II);

(3) making (III) a recombinant host cell comprising (II);

(4) a recombinant host cell produced by (III);

(5) an isolated antibody (IV) which specifically binds to (I);

(6) a recombinant host cell which expresses (I);

(7) preparation of (I);

(8) polypeptide prepared by the above method;

(9) the gene corresponding to the cDNA sequence of (I); and

(10) a product identified as a binding partner to (I), by contacting the polypeptide with the binding partner and determining whether it effects an activity of the polypeptide.

ACTIVITY - Cytostatic; Antiarthritic; Antirheumatic; Immunosuppressive; Antidiabetic; Neuroprotective; Nootropic; Antiallergic; Antiasthmatic; Antiparkinsonian; Cerebroprotective; Antiatherosclerotic; Antiinflammatory; Antianemic; Vasotropic; Ophthalmological; Nephrotropic; Gynecological; Antiulcer; Antiarrhythmic; Hepatotropic; Antithyroid; Vulnerary; Hemostatic; Protozoacide; Anti-HIV; Anticoagulant; Thrombolytic; Antibacterial; Virucide; Fungicide.

No supporting data is given.

MECHANISM OF ACTION - Vaccine; Gene therapy.

USE - (I) is useful for identifying a binding partner to the polypeptide. (I) and (II) are useful for preventing, treating or ameliorating a medical condition and also for diagnosing a pathological condition or susceptibility to the condition, by determining the presence or amount of expression of (I) or by detecting the presence or absence of mutation in (II). (II) is also useful for identifying an activity in a biological assay, by expressing it in a **cell**, isolating the supernatant, detecting the activity in a biological assay and identifying the protein in the supernatant having the activity (claimed). (I), (II) and (IV) are useful for diagnosis, prognosis, prevention and treatment of neurodegenerative disorders, e.g. Alzheimer's, Parkinson's diseases, retinitis pigmentosa, immune system disorders, such as cancer and autoimmune diseases, acquired or infectious, atherosclerosis, multiple sclerosis, insulin-dependent diabetes, chronic active hepatitis, AIDS, cirrhosis, allergic reactions and conditions such as asthma, inflammatory conditions, organ transplant rejection, infectious diseases including sarcoidosis, blood-related disorders, such as thrombosis, hemophilia, anemia, hyperproliferative disorders, renal disorders, e.g. acute kidney failure, inflammatory diseases of the kidney (glomerulonephritis), cardiovascular disorders, including peripheral artery disease such as limb

ischemia, arrhythmias, cerebrovascular disorders, respiratory disorders such as pneumonitis, lung cancer, tonsillitis, nonallergic rhinitis, pneumonia, Goodpasture's disease, endocrine disorders, including disorders and/or diseases of menstrual cycle, diabetes mellitus, Cushing's syndrome, thyroiditis, gastrointestinal disorders e.g. ulcers, such as pectic ulcers, **malabsorption** syndromes, inflammation of esophagus, dysphagia, and also muscular, reproductive disorders, and to enhance antiviral, antifungal, antibacterial and antiparasitic immune responses. The compounds also exhibit anti-angiogenic, chemotaxis activity, and also useful for epithelial **cell** proliferation, tissue regeneration and treating wound healing. (I) is useful for generating antibodies which are useful for purifying, detecting and targeting the polypeptides, including both in **vitro** and in **vivo** diagnostic and therapeutic methods, immunophenotyping of **cell** lines and biological samples and in immunoassays for qualitatively and quantitatively measuring levels of the polypeptides. (II) is useful for chromosomal identification, in gene therapy, forensic biology for detecting DNA sequences and as diagnostic probes for detecting presence of specific mRNA in a particular **cell** type, as molecular weight markers on Southern gels and as an **antigen** to elicit an immune response. (I) and (II) are useful in assays to test for one or more biological activities and for identifying agonists and antagonists of the polypeptides or polynucleotides which are useful for inhibiting **cell** growth and proliferation, prevent hypervascular diseases and to treat the above diseases. (I), (II) and the agonists or antagonists are also employed to prevent skin aging due to sun burn by stimulating keratinocyte growth, for preventing hair loss, as a food additive or preservative, to modulate mammalian characteristics and metabolism, to maintain organs before transplantation and to increase or decrease the differentiation or proliferation of embryonic stem cells.
Dwg.0/0

L4 ANSWER 9 OF 21 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN
ACCESSION NUMBER: 2002:212308 BIOSIS
DOCUMENT NUMBER: PREV200200212308
TITLE: Differential expression of CH809 by Candida albicans during oropharyngeal candidiasis and disseminated candidiasis.
AUTHOR(S): Cheng, S. [Reprint author]; Clancy, C. J. [Reprint author]; Checkley, M. [Reprint author]; Handfield, M. [Reprint author]; Hillman, J. D. [Reprint author]; Progulske-Fox, A. [Reprint author]; Lewin, A. S. [Reprint author]; Nguyen, M. H. [Reprint author]
CORPORATE SOURCE: University of Florida, Gainesville, FL, USA
SOURCE: Abstracts of the General Meeting of the American Society for Microbiology, (2001) Vol. 101, pp. 384. print.
Meeting Info.: 101st General Meeting of the American Society for Microbiology. Orlando, FL, USA. May 20-24, 2001. American Society of Microbiology.
ISSN: 1060-2011.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 27 Mar 2002
Last Updated on STN: 27 Mar 2002

AB We used a novel in **vivo** microbial expression system called In **Vivo** Induced **Antigen** Technology (IVIAT) to study the pathogenesis of oropharyngeal candidiasis (OPC). IVIAT uses anti-C. albicans antibodies from the sera of HIV-infected patients with OPC to identify antigens expressed by C. albicans during infection but not expressed during in vitro growth. We **adsorbed** the pooled sera from 24 HIV-infected patients with OPC with laboratory grown whole cells and **cell** lysates of a clinical C. albicans isolate, thereby

removing antibodies reactive against antigens expressed in **vitro**. We then used the adsorbed pooled sera to screen a C. albicans expression library by colony blot. We identified CH809, a gene that has not yet been reported in the Candida literature. This gene has a response regulator receiver domain that exhibits 88% identity at the amino acid level to the response regulator receiver domain of a kinase previously demonstrated to be a virulence factor for C. albicans. In order to study the expression of CH809, we isolated RNA from thrush samples and from in vitro grown C. albicans cells to perform RT-PCR. The C. albicans actin1 (act1) and elongation factor b1 (EFB1) genes were used as controls. The RT-PCR signal of CH809 from thrush samples was significantly higher than the product from in vitro grown C. albicans cells, whereas the RT-PCR signals of act1 and EFB1 were not different between the thrush samples and the in vitro grown cells. We next expressed the protein encoded by CH809 in E. coli, and performed Western analysis of the expressed protein using sera from 10 patients with OPC and 5 patients with disseminated C. albicans infections. The expressed protein was reactive with all sera obtained from patients with OPC but with none of the sera obtained from patients with disseminated candidiasis. Our preliminary data suggests that CH809 is expressed solely during OPC and not during disseminated candidiasis. The differential expression of this gene along with its role in pathogenesis of OPC is presently under further investigation.

L4 ANSWER 10 OF 21 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN

ACCESSION NUMBER: 2002:176530 BIOSIS
DOCUMENT NUMBER: PREV200200176530
TITLE: IVIAT screening of the entire Actinobacillus actinomycetemcomitans HK1651 genome for in vivo induced genes.
AUTHOR(S): Cao, S. [Reprint author]; Speigel, J. [Reprint author]; Progulske-Fox, A. [Reprint author]; Hillman, J. [Reprint author]; Handfield, M. [Reprint author]
CORPORATE SOURCE: University of Florida, Gainesville, FL, USA
SOURCE: Abstracts of the General Meeting of the American Society for Microbiology, (2001) Vol. 101, pp. 98. print.
Meeting Info.: 101st General Meeting of the American Society for Microbiology. Orlando, FL, USA. May 20-24, 2001. American Society for Microbiology.
ISSN: 1060-2011.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 6 Mar 2002
Last Updated on STN: 6 Mar 2002

AB We have applied In **Vivo** Induced **Antigen** Technology (IVIAT), a novel strategy for studying microbial pathogenesis and host interactions, to Actinobacillus actinomycetemcomitans (Aa), the etiologic agent of certain periodontal diseases including localized juvenile periodontitis. Sera from 20 periodontitis patients infected with Aa were pooled and repeatedly **adsorbed** with in **vitro** grown Aa HK1651 cells and **cell** lysates, leaving antibodies reactive with antigens expressed only in vivo. An expression library was constructed using 0.5 to 1.5kb Sau3A fragments of Aa HK1651 genomic DNA ligated into pET30abc. A second genomic expression library was constructed using hydrosheared DNA fragments to verify randomness and to ensure complete coverage of the entire chromosome. 200,000 clones, representing over 2.5X coverage of the Aa HK1651 genome, were probed with the adsorbed serum. All the clones initially identified by colony blot screening as being reactive with the adsorbed serum were confirmed using a quantitative dot blot assay. These confirmed clones were sequenced and analyzed using BLAST and MasterCatalog(R) software and were found to include 3 classes of

genes: 1) multiple in vivo induced genes have been identified that are known virulence-associated genes in Aa or in closely related organisms, 2) other in vivo induced genes have been identified that express proteins with known functions but have not previously been associated with pathogenesis, 3) various in vivo induced genes have been found that encode hypothetical proteins of unknown function. Immunofluorescence analysis of dental plaque and serological titration using sera from individual patients confirmed in vivo expression of a subset of these IVIAT clones. The in vivo induced antigens of Aa discovered using IVIAT are excellent candidates for use in diagnostic and therapeutic strategies, including rationale vaccine design. This work was supported by NIDCR grant DE13523 and iviGene Corporation.

L4 ANSWER 11 OF 21 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN

ACCESSION NUMBER: 2002:176528 BIOSIS
DOCUMENT NUMBER: PREV200200176528
TITLE: Probing for in vivo induced Porphyromonas gingivalis virulence genes using IVIAT.
AUTHOR(S): Song, Y. [Reprint author]; Kozarov, E.; Totten, K.; Skipper, A.; Ogen, A.; Handfield, M.; Hillman, J.; Progulske-Fox, A.
CORPORATE SOURCE: University of Chonbuk, Chonju, South Korea
SOURCE: Abstracts of the General Meeting of the American Society for Microbiology, (2001) Vol. 101, pp. 97. print.
Meeting Info.: 101st General Meeting of the American Society for Microbiology. Orlando, FL, USA. May 20-24, 2001. American Society for Microbiology.
ISSN: 1060-2011.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 6 Mar 2002
Last Updated on STN: 6 Mar 2002

AB The oral pathogen, P. gingivalis, is strongly associated with adult and refractory periodontitis and has been suggested to be involved in coronary heart disease. To identify P. gingivalis genes that are expressed during in **vivo** but not in vitro growth, a new approach, called In **Vivo** Induced **Antigen** Technology (IVIAT), was applied. Pooled sera from periodontitis patients were exhaustively **adsorbed** with in **vitro** grown whole P. gingivalis cells and **cell** extracts. The resulting serum containing only antibodies to in vivo induced (IVI) antigens was used to probe genomic expression libraries of P. gingivalis in E. coli using colony immunoscreening. Cloned DNA inserts from nine reactive clones were sequenced and analyzed to determine the cloned open reading frames (ORFs) likely to be responsible for expression of the IVI antigens. BLAST searches revealed one of the ORFs to have homology to a proteinase and another to a transcription repressor. Three ORFs were found to have no homology to sequences in the database and are thus likely previously unknown factors associated with virulence. The results of these studies which do not rely on animal models to mimic the growth of the pathogen in humans, are expected to improve our understanding of the pathogenic mechanisms employed by P. gingivalis by identifying virulence-associated genes that would not be found by conventional methods. These genes and their products are excellent candidates for therapeutic and diagnostic targets and for vaccine design.

L4 ANSWER 13 OF 21 MEDLINE on STN DUPLICATE 1

ACCESSION NUMBER: 1999012148 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9796056
TITLE: Enhanced immunogenicity of a recombinant genital warts vaccine adjuvanted with monophosphoryl lipid A.

AUTHOR: Thompson H S; Davies M L; Watts M J; Mann A E; Holding F P; O'Neill T; Beech J T; Thompson S J; Leesman G D; Ulrich J T
CORPORATE SOURCE: Cantab Pharmaceuticals Research Ltd, Cambridge, UK.
SOURCE: Vaccine, (1998 Dec) 16 (20) 1993-9.
Journal code: 8406899. ISSN: 0264-410X.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199812
ENTRY DATE: Entered STN: 19990115
Last Updated on STN: 20020926
Entered Medline: 19981231

AB The regression of genital warts is believed to be a T-cell-mediated immune effect. We have sought to enhance the immunogenicity of a therapeutic vaccine for the treatment of genital warts with the use of the adjuvant monophosphoryl lipid A (MPL-immunostimulant), a detoxified form of the lipopolysaccharide (LPS) of Salmonella minnesota R595. The comparative immunogenicity and reactogenicity of a recombinant human papillomavirus type 6 (HPV6) L2E7 fusion protein in either aqueous, oil-in-water emulsions or Alhydrogel formulations containing MPL was evaluated. We conclude that the simple addition of MPL to the L2E7 fusion protein already **adsorbed** onto Alhydrogel preferentially enhances **antigen** specific in **vitro** T-**cell** proliferative responses, IFN gamma production and in **vivo** delayed type hypersensitivity responses without increasing its reactogenicity.

L4 ANSWER 14 OF 21 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
ACCESSION NUMBER: 1997-042857 [04] WPIDS
CROSS REFERENCE: 1983-707055 [28]; 1988-056484 [08]; 1989-130047 [17]; 1990-305017 [40]; 1990-348485 [46]; 1992-096889 [12]; 1992-175125 [21]; 1992-200174 [24]; 1992-268664 [32]; 1992-331718 [40]; 1992-349203 [42]; 1993-018128 [02]; 1993-026900 [03]; 1993-076502 [09]; 1993-243234 [30]; 1994-263767 [32]; 1995-036113 [05]; 1995-366231 [47]; 1995-366385 [47]; 1996-187644 [19]; 1997-043114 [04]; 1997-051904 [05]; 1998-321465 [28]; 1998-332054 [29]; 1998-332055 [29]; 1998-332145 [29]; 1999-493494 [41]; 1999-610231 [52]; 2001-280989 [29]; 2002-040232 [05]; 2003-567445 [53]; 2004-601998 [58]
DOC. NO. CPI: C1997-013590
TITLE: Modified recombinant virus containing exogenous calicivirus DNA - useful in vaccines and for in vitro production of calicivirus antigens, for generation of therapeutic or diagnostic antibodies.
DERWENT CLASS: B04 C06 D16
INVENTOR(S): FISCHER, L; LEGROS, F; PAOLETTI, E
PATENT ASSIGNEE(S): (VIRO-N) VIROGENETICS CORP
COUNTRY COUNT: 22
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9639177	A1	19961212	(199704)*	EN	150
RW: AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL PT SE					
W: AU CA JP					
AU 9663152	A	19961224	(199715)		
EP 831899	A1	19980401	(199817)	EN	
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE					
JP 11511961	W	19991019	(200001)		160
US 5989561	A	19991123	(200002)		
AU 721567	B	20000706	(200038)		

AU 2000062470 A 20001214 (200103)#

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
WO 9639177	A1		WO 1996-IB721	19960603
AU 9663152	A		AU 1996-63152	19960603
EP 831899	A1		EP 1996-922179	19960603
			WO 1996-IB721	19960603
JP 11511961	W		WO 1996-IB721	19960603
			JP 1997-500280	19960603
US 5989561	A	Cont of	US 1991-666056	19910307
		CIP of	US 1991-713967	19910611
		Cont of	US 1992-847951	19920306
		CIP of	US 1993-36217	19930324
		CIP of	US 1993-105483	19930813
			US 1995-471025	19950606
AU 721567	B		AU 1996-63152	19960603
AU 2000062470	A	Div ex	AU 1996-63152	19960603
			AU 2000-62470	20001004

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9663152	A	Based on	WO 9639177
EP 831899	A1	Based on	WO 9639177
JP 11511961	W	Based on	WO 9639177
US 5989561	A	CIP of	US 5364773
		CIP of	US 5494807
AU 721567	B	Add to	AU 701781
		Previous Publ.	AU 9663152
		Based on	WO 9639177
AU 2000062470	A	Div ex	AU 721567

PRIORITY APPLN. INFO: US 1995-471025 19950606; US
1991-666056 19910307; US
1991-713967 19910611; US
1992-847951 19920306; US
1993-36217 19930324; US
1993-105483 19930813; AU
2000-62470 20001004

AN 1997-042857 [04] WPIDS

CR 1983-707055 [28]; 1988-056484 [08]; 1989-130047 [17]; 1990-305017 [40]; 1990-348485 [46]; 1992-096889 [12]; 1992-175125 [21]; 1992-200174 [24]; 1992-268664 [32]; 1992-331718 [40]; 1992-349203 [42]; 1993-018128 [02]; 1993-026900 [03]; 1993-076502 [09]; 1993-243234 [30]; 1994-263767 [32]; 1995-036113 [05]; 1995-366231 [47]; 1995-366385 [47]; 1996-187644 [19]; 1997-043114 [04]; 1997-051904 [05]; 1998-321465 [28]; 1998-332054 [29]; 1998-332055 [29]; 1998-332145 [29]; 1999-493494 [41]; 1999-610231 [52]; 2001-280989 [29]; 2002-040232 [05]; 2003-567445 [53]; 2004-601998 [58]

AB WO 9639177 A UPAB: 20041223

A novel modified recombinant virus (A) has virus-encoded genetic functions inactivated so that virulence is attenuated but efficacy is retained, and includes exogenous DNA (I) encoding at least one calicivirus **epitope** in a non-essential region of the viral genome. Also new are: (1) calicivirus antigens (Ag) prepared from in vitro expression of (A); (2) antibodies (Ab) elicited by in **vivo** expression of Ag from (A) or by admin. of Ag prepared in vitro; and (3) probes and primers derived from the DNA of (A).

USE - (A) are used as vaccines to generate a immunological response

(claimed) against calicivirus, especially rabbit haemorrhagic disease virus (RHDV), in vivo, and to express a gene product (such as an Ag) by in **vitro cell culture** (claimed). They can also be used to analyse the relative importance of humoral and **cell** -mediated responses in protection against calicivirus infection. Ab are useful as diagnostic reagents for detecting virus, infected cells or Ag, also for treatment and prevention of calicivirus disease, or **immunoadsorption** separation of virus. Ag are used to detect or generate Ab.

ADVANTAGE - (A) are safer than known recombinant poxvirus vaccines and allow Ag to be produced in vitro without risk of infection.
Dwg.0/14

L4 ANSWER 15 OF 21 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1996:490072 CAPLUS
DOCUMENT NUMBER: 125:140133
TITLE: Liver cell uptake and degradation of soluble immunoglobulin G immune complexes in vivo and in vitro in rats
AUTHOR(S): Johansson, Anders G.; Lovdal, Torunn; Magnusson, Karl-Eric; Berg, Trond; Skogh, Thomas
CORPORATE SOURCE: Departments Medical Microbiology, Linkoping University, Linkoeping, S-58185, Swed.
SOURCE: Hepatology (Philadelphia) (1996), 24(1), 169-175
CODEN: HPTLD9; ISSN: 0270-9139
PUBLISHER: Saunders
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Immune complexes were formed between dinitrophenylated human serum albumin (DNP-HSA) and polyclonal rabbit IgG anti-DNP antibodies at antibody excess. The antigen was labeled with isotope (125I-tyramine-cellobiose) or fluorochrome, (6-[fluorescein-5-(and-6)-carboxamido] hexanoic-acid, succinimidyl ester). The radiolabeled antigen, native or antibody complexed, was given i.v. to rats. Radioactivity was measured in various organs at 1 h following injection. The liver was the main site for removal of the antigen as well as of the immune complexes. Within the liver, immune complexes were mainly associated with nonparenchymal liver cells, the total recovery from Kupffer cells being about 10 times greater than from the liver endothelial cells. The uncomplexed radiolabeled antigen was readily degraded by both cell types. After IgG complexing, the degradation decreased, both in Kupffer cells and in liver endothelial cells. In vitro expts. with isolated liver cells, showed that IgG complexing increased antigen uptake to about the same extent in Kupffer cells and in liver endothelial cells. The degradation of both **antigen** and immune complexes was less efficient in vitro than in **vivo**. Immune complex uptake in vitro was shown also by confocal fluorescence microscopy in Kupffer cells and in liver endothelial cells. Also in vitro, only minor uptake was found in the hepatocytes. We conclude that both liver endothelial cells and Kupffer cells are involved in the hepatic handling of soluble IgG immune complexes, but we found no evidence for substantial uptake by hepatocytes.

L4 ANSWER 16 OF 21 WPIDS COPYRIGHT 2005 THE THOMSON CORP on STN
ACCESSION NUMBER: 1991-014911 [02] WPIDS
CROSS REFERENCE: 1990-224385 [29]
DOC. NO. CPI: C1991-006446
TITLE: Hindered linking agents - are substd. 2-imino thiolane hydrohalide(s), used to form di sulphide linkages.
DERWENT CLASS: B03 B04
INVENTOR(S): CARROLL, S F; GOFF, D A
PATENT ASSIGNEE(S): (XOMA) XOMA CORP
COUNTRY COUNT: 2

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
ZA 9000048	A	19901031	(199102)*		
US 5093475	A	19920303	(199212)		15
US 5183904	A	19930202	(199308)		13

APPLICATION DETAILS:

PATENT NO	KIND		APPLICATION	DATE
ZA 9000048	A		ZA 1990-48	19900104
US 5093475	A		US 1989-454576	19891221
US 5183904	A	CIP of	US 1988-288586	19881222
		Div ex	US 1989-454576	19891221
			US 1991-796048	19911120

FILING DETAILS:

PATENT NO	KIND		PATENT NO
US 5183904	A	Div ex	US 5093475

PRIORITY APPLN. INFO: US 1989-454576 19891221
AN 1991-014911 [02] WPIDS
CR 1990-224385 [29]
AB ZA 9000048 A UPAB: 19950602
2-Imino-thiolane derivs. of formula (I) are new: X = halo; R1 = COOR5, halo, NO2, 1-8C haloalkyl, 1-8C alkoxy, 2-8C alkenyl or 2-8C alkynyl (all opt. substd. by halo), 3-8C cycloalkyl, aryl or heterocycle (both opt. substd. by 1-3 of halo, amino or opt. halo-substd. 1-8C alkyl or alkoxy); each of R2,R3 and R4 = H or as for R1; R1+R2 can together be 2-5C alkylene, opt. substd. by 1-5 1-4C alkyl; or R1 or R2 plus R3 are together 1-5C alkylene, opt. substd. by 1-5 1-4C alkyl); R5 = H or 1-8C alkyl. Also new are conjugates (especially immunotoxins) having at least one linkage of formula (II) and consisting of a first component (A, especially cytotoxic agent) coupled to a second component (B, especially antigen-reactive agent) at an amino or carbohydrate gp. native to (B).

USE/ADVANTAGE - (I) (and the cpds. with R1 = lower alkyl and R2-R4 = H or alkyl, known as intermediates for herbicides and insecticides) are crosslinking agents for linking two proteins with site-specific disulphide bond of controlled stability. Since (I) are water soluble, conjugation can be carried out under aqueous conditions, avoiding damage to proteins caused by organic solvents. The (I) - derived linkages are more stable (against reduction enzymes and disulphide exchange) than those derived from unsubstd. 2-iminothiolanes. The immunotoxins are useful in therapy for targetting specific cells. (Provisional Basic - previously advised in week 9048) @(44pp Dwg.No.0/2)@
0/2
ABEQ US 5093475 A UPAB: 19930928
Covalent linking of 2 species comprises (a) reacting 1 or both species with a substd. 2-iminothiolane hydrohalide crosslinking agent of formula (I), (b) desalting to remove reagent and (c) mixing the 2 species together. In (I), X is halo; R1 is COOR5, halo, NO2, opt. halogenated 1-8C alkyl, 1-8C alkoxy, 2-8C alkenyl or 2-8C alkynyl, 3-8C cycloalkyl, or aryl or heterocyclic gp. opt. substd. with 1-3 of halo, NH2, opt. halogenated 1-8C alkoxy or opt. halogenated alkyl; R2, R3 and R4 are each H or R1 or R1 and R2 together form a 2-5C alkylene bridge opt. substd. with 1-5 1-4C alkyl gps. or R1 and R2 together with R3 form a 1-5C alkylene bridge opt. substd. with 1-5 1-4C alkyl gps.

One of the species is pref. a polypeptide and the other is a protein,

esp. a cytotoxic agent and antigen reactive agent. The cytotoxic agent is a ribosomal inactivating protein and the antigen reactive agent is an immunoglobulin.

ADVANTAGE - UV absorbing cpd. (I) allows direct measurement of its reaction with a protein. The linking reaction may be controlled.

ABEQ US 5183904 A UPAB: 19930928

New substd. 2-iminothiolane hydrohalide have formula (I). X is halo; R1 is COOR5, halo, nitro, halogenated 1-8C alkyl, unsubstd. or halogenated 1-8C alkoxy; unsubstd. or halogenated 1-8C alkenyl, unsubstd. 3-8C cycloalkyl, aryl (opt. substd. by 1-3 substits. e.g. halo, amino, unsubstd. or halogenated 1-8C alkyl or alkoxy) or heterocycle (opt. substd. by 1-3 substits. as above); R2-R4 are independently H or selected values of R1 or R1 and R2 together form 2-5C alkylene bridge opt. substd. by 1-5, 1-4C alkyl; and R5 is H or 1-8C alkyl. Also R1 or R2 together with R3 form a 1-5C alkylene bridge opt. substd. as above.

USE/ADVANTAGE - (I) are used as linking agents partic. for synthesis of immunotoxins comprising cytoxic agents which is fatal to a **cell** when **absorbed** and an **antigen**-reactive agent for delivering the toxic agent to the partic **cell**. (I) are advantageous as linking agents because they exhibit increased stability to redn. in **vitro** and in **vivo** due to sterically hindered gps. of R1 to R4.

0/2

L4 ANSWER 17 OF 21 MEDLINE on STN DUPLICATE 2

ACCESSION NUMBER: 85235599 MEDLINE
DOCUMENT NUMBER: PubMed ID: 4008930
TITLE: The serologic response to Meth A sarcoma vaccines after cyclophosphamide treatment is additionally increased by various adjuvants.
AUTHOR: Livingston P O; Jones M; Deleo A B; Oettgen H F; Old L J
CONTRACT NUMBER: CA-28461 (NCI)
SOURCE: Journal of immunology (Baltimore, Md. : 1950), (1985 Aug) 135 (2) 1505-9.
Journal code: 2985117R. ISSN: 0022-1767.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198508
ENTRY DATE: Entered STN: 19900320
Last Updated on STN: 19970203
Entered Medline: 19850819

AB We have shown previously that the serologic response of BALB/c mice to immunization with BALB/c sarcoma Meth A cells can be more effectively augmented by pretreatment with cyclophosphamide (Cy) than by the use of adjuvants. The serologic response was directed against a highly restricted cell surface antigen, closely related to or identical with the unique transplantation antigen characteristic for this tumor. In this paper, we report the results of our attempts to obtain additional augmentation by using Cy and adjuvants together. For these studies, the optimal Cy dose, interval between Cy and vaccine administration, and vaccine cell number were determined. Mice were injected with Cy 25 mg/kg i.p., and 3 days later, with viable irradiated (10,000 rad) Meth A cells subcutaneously, under conditions in which only few mice produced antibody. Sera were tested for antibody with reactivity against Meth A by complement dependent cytotoxicity (CDCX), which predominantly detects IgM, and by the protein A (PA) and anti-IgG assays, which detect IgG. Of the various adjuvants tested, only monophosphoryl lipid A (MPLA) and CP20,961 resulted in significantly increased titers of reactivity in both the CDCX and PA assays over that obtained when using Cy alone. Although the mean titers observed with CDCX ranged between 1/160 and 1/320, no titer above 1/40 was

observed with the PA assay. The specificity of the CDCX reactivity detected by the assay for the Meth A **antigen** was ascertained by **absorption** analysis of selected sera by using a panel of BALB/c spleen and tumor **cell** lines grown in **vitro** or in **vivo**. PA titers were too low to permit absorption analysis, and the titers obtained in the anti-IgG assay were lower still. Attempts to augment the anti-Meth A IgG response or to convert the IgM response to IgG were unsuccessful. The combined approach described here (i.e., vaccination with irradiated syngeneic tumor cells plus MPLA in Cy-pretreated mice) was also shown to be effective in augmenting the serologic response against two additional murine leukemia virus-negative sarcomas that are known to be less immunogenic, CMS4 and CMS5. It appears, therefore, that this combined approach may be applicable to stimulating serologic responses against a variety of tumor cell surface antigens.

L4 ANSWER 18 OF 21 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation on STN DUPLICATE 3

ACCESSION NUMBER: 1981:147237 BIOSIS
DOCUMENT NUMBER: PREV198171017229; BA71:17229
TITLE: MURINE T CELL MEDIATED CYTO TOXICITY AGAINST SYNGENEIC AND ALLOGENEIC CELL LINES INDUCED BY FETAL CALF SERUM.
AUTHOR(S): THORN R M [Reprint author]
CORPORATE SOURCE: CANCER BIOL PROGRAM, NATL CANCER INST FREDERICK CANCER RES CENT, FREDERICK, MD 21701, USA
SOURCE: Cellular Immunology, (1980) Vol. 54, No. 1, pp. 203-214. CODEN: CLIMB8. ISSN: 0008-8749.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH

AB Mouse spleen cells from normal animals developed easily measurable cytotoxicity against various cell lines [mouse UV light-induced fibrosarcoma ZC16, 1591, 1316, 112 and 2237 cells; mouse methylcholanthrene-induced fibrosarcoma 3152 and 113 cells; mouse Maloney sarcoma virus-induced YAC cells; mouse benzanthracene-induced lymphoma EL4 cells; mouse fibroblast BS cells] when cultured in vitro without deliberate sensitization. Cytotoxicity, measured by a 3 h 51Cr-release assay, was maximum on days 3 and 4 of culture and was dependent on the presence of fetal calf serum. Cell recovery or blastogenesis did not correlate with the amount of cytotoxicity generated. Nylon wool **adsorption** of effector cells **cultured** 3 days had only a marginal effect on cytotoxicity; cytolysis was markedly reduced (but not totally eliminated) by treatment with anti-T **cell** serum and complement. When target cells were in relative excess to effector cells, 51Cr release was proportional to effector cell number and proceeded for at least 22 h. The cytotoxicity was not tumor or H-2 specific. Targets without known C-type viral antigens (gp71) were killed as readily as those with easily measurable viral antigens. Nontumorigenic fibroblasts were lysed; concanavalin A-induced blast cells were not. Cytotoxicity was not augmented in cultures of spleen cells from mice injected with fetal calf serum or with tumor fragments exposed to fetal calf serum. Mercaptoethanol was not necessary for the generation of cytotoxic activity; T cells and Sephadex G-10 or nylon wool-adherent cells were necessary and the function of the adherent cell could not be replaced by mercaptoethanol. Removal of plastic adherent cells had no effect. Fetal calf serum retained its activity when heated for 45 min at 56° C or when dialyzed. Dilution and reconcentration by Amicon filtration revealed that the mass of the active material was between 30,000 and 100,000 daltons. The early appearance, transient nature and nonspecificity of this cytotoxicity distinguish it from antigen-specific reactions. The effector's stability at 37° C and its relatively easily detectable T cell markers distinguish it from natural killer and

cytotoxic cells. This activity is like lectin-induced cytotoxicity but differs because allogeneic blast cells are not lysed. The observed cytotoxic activity may be of in **vivo** relevance (vis-a-vis natural killer cells) or, more likely, an in vitro expression of a stage of cell differentiation that T cells may normally pass through during their response to **antigen**.

L4 ANSWER 19 OF 21 MEDLINE on STN DUPLICATE 4
ACCESSION NUMBER: 78111471 MEDLINE
DOCUMENT NUMBER: PubMed ID: 75266
TITLE: Tumor-specific antigens on rat liver cells transformed in vitro by chemical carcinogens.
AUTHOR: Yokota T; Sizaret P; Martel N
SOURCE: Journal of the National Cancer Institute, (1978 Jan) 60 (1) 125-9.
Journal code: 7503089. ISSN: 0027-8874.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197804
ENTRY DATE: Entered STN: 19900314
Last Updated on STN: 19900314
Entered Medline: 19780426

AB With the use of membrane immunofluorescence and xenogeneic antisera, tumor-specific membrane antigens were detected on rat epithelial-like liver cells transformed in vitro by chemical carcinogens. These antigens were not detected in 10-, 15-, and 19-day rat fetuses. Xenogeneic antisera were produced in rabbits by immunization of the rabbits with cultivated BD rat liver cells transformed by dimethylnitrosamine or N-methyl-N'-nitro-N-nitrosoguanidine. The specific antisera against tumor-associated **antigen**(s) were obtained by in **vivo** **absorption** in syngeneic male rats and by in **vitro** **absorption** with various **cell** lines. One tumor-specific individual antigen and two tumor-specific cross-reacting antigens were shown to be present on the surface of chemically and/or spontaneously transformed rat liver cell lines. They were not detected on liver and spleen cells of normal BD adult rats, on fetal liver cells, or on liver and intestinal carcinoma cells of Wistar rats. Sera from multiparous pregnant rats had no antibodies against these tumor antigens (although they reacted with fetal cells).

L4 ANSWER 20 OF 21 MEDLINE on STN DUPLICATE 5
ACCESSION NUMBER: 77045326 MEDLINE
DOCUMENT NUMBER: PubMed ID: 990168
TITLE: Hypersensitivity to bacteria in eczema. IV. Cytotoxic effect of antibacterial antibody on skin cells acquiring bacterial antigens.
AUTHOR: Parish W E; Welbourn E; Champion R H
SOURCE: British journal of dermatology, (1976 Nov) 95 (5) 493-506.
Journal code: 0004041. ISSN: 0007-0963.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197701
ENTRY DATE: Entered STN: 19900313
Last Updated on STN: 19900313
Entered Medline: 19770129

AB The sera of persons with generalized eczema (Whitfield-type) or with disseminated nummular eczema were examined for complement-activating antibacterial antibodies to test the hypothesis that some eczematous

change results from an antibody-mediated cytotoxic reaction. Bacteria dying in the stratum corneum release soluble antigens, some of which diffuse into the stratum Malpighii and become firmly adsorbed to the epidermal cells. Antibacterial antibody and complement diffusing into the epidermis react with the antigens acquired by the cells and may induce vacuolation or lysis. Phenol-extracted and freeze-press-extracted antigens (both containing teichoic acids) from Staphylococcus aureus and a micrococcus (Baird-Parker types SI and MI respectively) are adsorbed by monolayers of human skin, embryo or amnion. Cells acquiring the antigen(s) are severely damaged when treated with sera containing the appropriate antibacterial antibodies and complement. IgM complement-fixing antibody appears to be much more cytotoxic in this test than IgG. The cytotoxic activity of a serum is specific for the acquired bacterial antigen and appears to depend on a sufficient concentration of the effective antibody, and not on the presence of antibodies with special properties. Explants of full thickness skin treated with bacterial antigen extracts were unharmed by the antibodies that were cytotoxic for monolayers of skin cells treated with the same antigens. The in **vitro** cytotoxic test should represent a potential in **vivo** cytotoxic phenomenon, because skin **cell** monolayers from two patients **adsorbed** bacterial **antigen** prepared from cultures obtained from the same patients, and were damaged by autologous serum containing anti-staphylococcal antibody and complement. It seems probable that this may be an aggravating but not necessarily an initiating factor in many cases of eczema.

L4 ANSWER 21 OF 21 EMBASE on STN

ACCESSION NUMBER: 77061284 EMBASE
DOCUMENT NUMBER: 1977061284
TITLE: Preparation and properties of antisera directed against antigens of the P 815 mastocytoma cell not shared by its syngeneic host, the DBA/2 mouse.
AUTHOR: Bertschmann M.; Clemetson K.J.; Luscher E.F.
CORPORATE SOURCE: Theodor Kocher Inst., Univ. Berne, Switzerland
SOURCE: European Journal of Cancer and Clinical Oncology, (1976) Vol. 12, No. 4, pp. 255-262.
CODEN: EJCAAH
DOCUMENT TYPE: Journal
FILE SEGMENT: 016 Cancer
026 Immunology, Serology and Transplantation
025 Hematology
LANGUAGE: English

AB P 815 mastocytoma cell, upon intradermal injection, induce an immune response in the syngeneic host, the DBA/2 mouse, in the course of which the tumor may be rejected due to the presence of immunocompetent cells. No evidence for the production of humoral cytotoxic antibody has as yet been found. However, such antibodies, directed specifically against **antigen**(s) of the P 815 cell can be produced by immunizing C3H mice or sheep and by subsequent in **vivo** absorption of the allo or xeno antisera in the DBA/2 mouse. By in **vitro** **absorption** of such in vivo **absorbed** anti P 815 antisera with P 815 cells or DBA/2 spleen cells, respectively, it can be demonstrated that the specificity of the in vivo **absorbed** antiserum is exclusively determined by antigenic determinants of the tumor **cell**. As expected, immunization of the C3H mouse with normal DBA/2 tissue induces an antiserum, which, after in vivo absorption in the DBA/2 mouse has no cytotoxic effect against the P 815 cell. The injection of the in vivo absorbed anti P 815 antiserum in DBA/2 mice which are challenged with P 815 cells, leads to a delay in the development of the tumor but not to a complete depression of tumor growth. Full inhibition of tumor growth was achieved, however, when complement (guinea pig) was

injected at the same time. The possible relationship of the P 815 specific antigen(s) inducing a humoral response and the antigen(s) inducing rejection in the syngeneic system is still unclear.

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L1          3028 (CULTURE OR CULTURED OR VITRO) (S) CELL (S) (?ADSORB OR ?ADSORB
L2         26612 VIVO (S) (ANTIGEN OR EPITOPE)
L3            32 L1 AND L2
L4            21 DUP REM L3 (11 DUPLICATES REMOVED)

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